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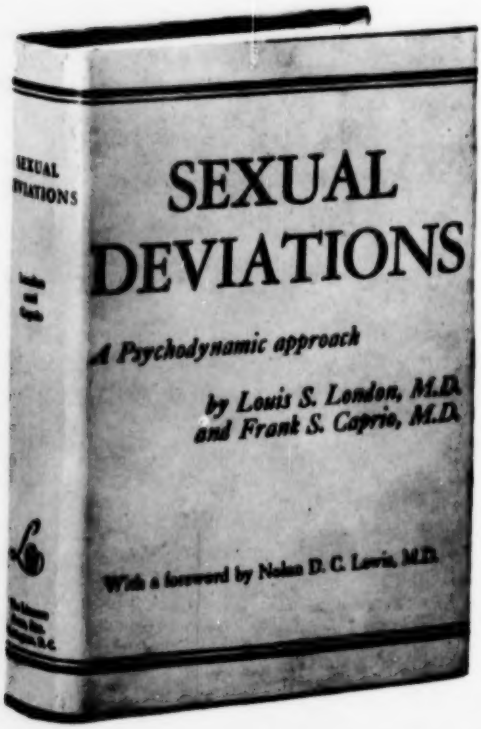
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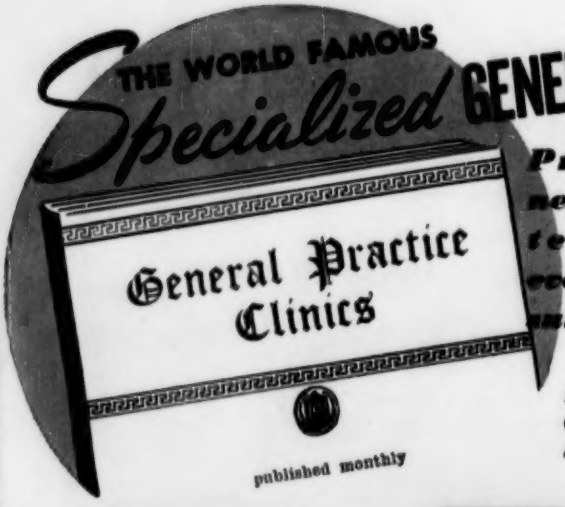

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Seasonal and Perennial Hayfever and Asthma in Children

A Practical Analysis on Treatment

ALEXANDER STERLING, M.D.*

This paper is a summary of a study of 220 children of a total of 384 from the ages of 1 to 14, who have called for treatment in the last 15 years (1930–1945). Of this series 164 are not included in this analysis because they did not have a complete study. There were 100 boys and 120 girls.

For the sake of clearness I would like to use the following sub-divisions: (1) 32 patients or 14.5% with *perennial rhinitis* due to inhalants, dust and bacteria; (2) 23 patients or 10.5 % with *perennial asthma* due to inhalants, dust and bacteria; (3) 17 patients or 7.7% with *perennial rhinitis and asthma* due to inhalants, dust and bacteria; (4) 19 patients or 8.6 % with *seasonal hayfever* due to single or mutiple seasonal pollen; (5) 57 patients or 25.9% with *seasonal hayfever* due to single or multiple seasonal pollen, but continuing throughout the year due to other causes such as dust and bacteria, inhalants and, occasionally, foods; (6) 36 patients or 16.4% with *seasonal hayfever and asthma* due to single or multiple pollen, but their attacks were confined to the seasons only; (7) 36 patients or 16.4% with *seasonal hayfever and asthma* due to single or multiple pollen, but continuing throughout the year due to other causes, such as inhalants, dust, recurrent upper respiratory infections and, occasionally, foods.

The 220 patients had complete allergic studies. It is of interest to note that we had only 10 patients with food sensitivity. While it was a major factor in the production of hayfever and asthma, it was not the only factor in each instance. It was always associated with other responsible etiologic factors such as pollen and dust. The foods were eggs, milk, orange juice, some fishes, some meats and wheat.

Patients subject to a single seasonal pollen are the easiest to cure. It makes little difference whether their sensitivity is due to trees, grass, ragweed, etc. It is more difficult to obtain favorable results when subject to more than one pollen. Pollen desensitization[1] *per se* is the same whether the patient is suffering from seasonal *hayfever* alone or from seasonal *hayfever and asthma* combined. The most difficult patients are those who are suffering from perennial hayfever and asthma in the warm months due to pollen, and in the winter months when their symptoms are initiated and aggravated by frequent colds and infection. *One must not forget that patients whose hayfever and asthma can be initiated by colds and infections may be sick in the pollen season, when their sickness is not due to pollen.*

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These patients present the greatest problem in the differential diagnosis. If symptoms develop at the height of the seasons, it takes a great deal of examination and evaluation to decide when the acute attack of hayfever and asthma is *not* the result of pollen contact but the result of an acute cold and infection. When the weather is usually warm the average person is expected to be free from chronic common colds at the time. Colds or infections are the last thing in the mind of the physician.

Individuals susceptible to bacterial infections and common colds can break out with acute upper and lower respiratory infections in and out of pollen season. Thus, while under treatment for pollen, if respiratory symptoms appear shortly before, after or during the course of the pollen season, they present a severe problem for the physician. First, is it due to overdosing of pollen, and if so, is it necessary to decrease the pollen strength? Secondly, is it due to not enough pollen protection and should the pollen injections be more frequent or the concentration of the pollen dilutions increased? Thirdly, is it due to the patient's susceptibility to bacterial infections and common colds and if so, is this present acute attack due to a cold and not pollen contact? Has the patient a history of having had colds throughout the year? Does examination of the patient's nose and throat show redness and congestion, as well as tender cervical glands, especially in the presence of a faster pulse and fever? Then the diagnosis is clear. The injections for pollen desensitization should be stopped for the duration of acute illness and the patient should be treated on general principles for a cold.

Regardless of single or multiple states of sensitivities present in the patients: 60% were well after six months; 10% were well after the first year of treatment; 10% were well after 18 months of treatment; 10% were well after two years of treatment; 7%, after two years of treatment, were still having recurrent attacks and needed further studies and further evaluations of their etiologic factors, such as re-checking or re-testing. However, they were well after three to three and a half or four years of treatment; 3% did not respond to treatment no matter what form of desensitization was carried out. They had recurrent attacks of rhinitis, bronchitis and attacks of asthma. It was impossible to regulate them or determine causes which could be responsible for their recurring attacks.

For years it has been suggested by allergists that patients who call for treatment with hayfever and asthma symptoms should have a complete allergy study.[2] Still many who called at our office gave a history of having had only partial studies. Incomplete study is usually a disappointment to the physician as well as the patient and should be avoided. Immediate as well as long-range plans must be made for the general treatment of the patient as well as for the specific allergic rhinitis or asthma. Every phase of their physical deficiency should be corrected. Detailed environmental factors should be regulated. Their associations at home and in school must be evaluated and studied before perfect results can be obtained. A complete testing, which is imperative in every case, is only a

guide and must be checked clinically. After every recurrent attack, the patient should undergo a further study to locate the causes which could be responsible for the recurring allergic manifestations. He should be referred to a nose and throat man for guidance and treatment. Some have to have a change of environment as a test. This does not mean that a Philadelphian should go away to distant places such as Florida or Arizona "or vice versa," but to a different home in another neighborhood in the same city, or to an adjacent country or seashore environment. It should entail a radical change from the patient's former home to the completely new atmosphere selected. It should include different types of furniture, bedding, etc. The patient's nose and throat and chest should be examined before and after his vacation to determine if any benefits were derived from the environmental changes.

Browsing in a medical library in Guy's Hospital, London, in January, 1930, I came across a medical report which is of interest. A case report by a member of the Royal College of Physicians and Surgeons at a meeting in 1780 reads as follows: "He is a personal physician to a Member of Parliament who lived on an estate on the outskirts of London. The patient is of good habits and fine manners and in perfect health otherwise. Every year in May and June he has been suffering from attacks of shortness of breath, swollen, blocked and running nose, watering and swollen eyes. When he reached London for a meeting with Parliament, he was a new man. All his difficulty in breathing had subsided, his nose and eyes had been more comfortable and he was perfectly well as long as he stayed in London. The symptoms always recurred upon his return to his estate. This experience has taken place on three consecutive years. In the last year while there were no Parliament meetings in the month of May, he made arrangements to reside somewhere in the vicinity where the Parliament meetings were held which was almost in the slums of London. To our greatest surprise, in 24 to 36 hours, the patient had completely recovered from all the complaints."

The doctor could not give any explanation except that he wanted to register his three or four years of observation to the Royal College of Physicians. He questioned the theory of "miasms" (bad air) as a responsible factor for asthma. A doctor who discussed the case ridiculed this observation. "How could the slums of London keep the patient free from obnoxious symptoms?"

This case report was forgotten, as were Fleming's experiments with penicillin in our present generation. It was not brought to light until 1819, almost 40 years later, when John Bostock described before the Royal Medical and Chirurgical Society of London his own "Case of a Periodical Affliction of the Eyes and Chest" which we now know are the classical symptoms of grass hayfever and asthma. This published incident is an illustration of what change of residence may do to a patient even though the real responsible etiologic factors may not be known.

All possible *precipitating factors* should be discussed with the patient,

parents or guardians. One often finds the following responsible for the recurrences: acute colds, exhaustion from running, wrestling or playing ball, unduly prolonged drives in old upholstered automobiles, long walks through the country, or a visit to farms where contact may be made with sensitive factors.

Sometimes the recurrences are due to overdosage with antigens used in the treatment. When in doubt, it is best to fall back to much weaker dilutions and do another work-up of the patient. Checking by intradermal testing is occasionally misleading but when it is done often—once in several weeks—the state of the patient's intradermal response may be learned and his whealing reaction may be read correctly. Most of the antigenic dilutions must be made fresh every two weeks. When checking, the dose should not be advanced larger than that which produces a whealing the size of a penny to a nickel.

The majority of the patients studied for this analysis immediately responded to treatment and became perfectly free from symptoms. Treatment was continued from at least one to two years. Some patients did not improve with the first outlined course of treatment. While they had recurrences, their attacks were less frequent and of much shorter duration. They responded to symptomatic treatment more promptly as compared with response prior to the institution of desensitization.

The benefits obtained from injections of antigens do not depend upon hurriedly working up the patient to stronger concentrations. It depends upon giving the patient *weak* dilutions *at rather frequent intervals* and prolonged for months and years. In some instances the same dilution may have to be continued two or three times a week for several months.

"Can asthma and hayfever be cured?" is always asked by the patient. From this analysis, demonstrating perfect results, the answer to the question is as follows: "Your cure depends on the following: (1) a complete allergic study; (2) your cooperation in removing all the allergenic factors from your environment which may be suggested by the allergic study; (3) your coming frequently for injections to be desensitized for all the responsible factors in your case."

The frequency of injections depends on how many antigenic dilutions will be required. The minimum number of treatments would be from three to four times a week. For the patients who cannot call that often, someone of the family is instructed about injections. In either case, it is necessary to come at least once a week at the beginning to check up the doses. After several months the check-ups are once in two weeks. As the patient improves, less and less frequency is required for a check-up. When patients are cooperating and carrying out all the necessary treatment and preventive measures of environment, food, etc., they usually get well within one to two years. This is illustrated by the following cases:

A girl 9 years of age was seen on October 15, 1945, with a severe attack of asthma and bronchitis. It began in July and kept up throughout the

months of August, September and October. She has had similar experiences for the last three years.

C. C.—Coughing and wheezing were worse for the last two or three weeks even though the pollen season was over. There was no sneezing nor redness or itching of the eyes. For the last three weeks she has had severe nocturnal attacks of asthma. The family physician had to be called in once or twice during the night to relieve her attacks with adrenalin.

P. H.—Always subject to colds in the winter followed by bronchitis, wheezing and asthma. She had the usual childhood diseases. Her tonsils and adenoids were removed at four. Upon examination she had a fever of 101° F., pulse rate of 120, acute pharyngitis, bronchitis and wheezing. The family physician was advised to discontinue injections of adrenalin,[3] to apply mustard plasters to the chest, to administer aspirin, phenacetin, caffeine citrate, sulfadiazine in the proper doses, and daily injections of penicillin.[4] In four days the patient was better from the bronchitis and asthmatic attacks.

Comment: The patient, subject to colds shortly before or after pollen seasons, was treated as a continuation case of pollinosis, and more pollen therapy was instituted year after year. Pollen injections were given by the family physician, under supervision of an allergist, but they lost sight of the fact that bacterial infection and colds held an important role as an etiologic factor. We advised the following treatment: an autogenous vaccine, including smears from the nose, throat and sputum, a dust extract from her home environment and smaller doses of mixed ragweed pollen. The patient had no more recurring attacks in or out of season for the past three years.

The second patient, a boy aged 10, was seen in May, 1945.

C. C.—Sneezing and coughing from April to September for the last five or six years. He was better but not well the rest of the year. He was unable to sleep at nights on account of nasal obstruction and precipitating severe attacks of sneezing and coughing.

P. H.—Measles at age 5, chickenpox at 6, and appendectomy at 9. He has had four attacks of acute otitis media. He lost his hearing on the left side as a result of the frequent attacks of rhinitis, colds and otitis media. There was no itching of the eyes, nose and throat. He has had one attack of urticaria and angioneurotic edema of face and body. He has had coughing spells after exercise. Bathing, at home or in pools, would always induce sneezing spells.

F. H.—His grandmother on mother's side had asthma and rheumatism. An aunt on his mother's side had hives and nasal obstruction. A cousin on his mother's side had hayfever and asthma. He was tested for pollen, foods, inhalants, bacteria and dust, and was found to be positive to grass, plantain and ragweed, each plus two; dust, plus one; bacterial proteins, such as Staphylococcus, Streptococcus, Micrococcus tetragenus,

and pneumococcus, each plus one. He was positive to eggs, beef, cucumber, paprika, corn and spinach.

We instituted the following treatment: ragweed pollen, starting with 1:100,000 dilution; a mixture of grass and plantain pollen, dilution of 1:100,000; dust, 1:200, and with a special vaccine made from his nose and throat, dilution 1:50. The patient's mother was instructed in the use of the hypodermic needle. Injections were given six times a week: Monday, Wednesday and Friday, ragweed pollen and dust; Tuesday, Thursday and Saturday, grass and plantain mixture and the autogenous vaccine. The following spring, summer and fall he was free from symptoms. After the first year of treatment the hearing was better by 50%. The concentration of all antigenic dilutions was gradually increased. He was discharged well after two years.

The third patient, a boy 10 years of age, was seen on October 15, 1941.

C. C.—Fall hayfever and asthma for the last four seasons.

P. H.—Measles at age 9, T and A at 3. He was subject to colds with nasal obstruction throughout the year which was worse at night and on going to bed, when he had coughing spells accompanied with greenish-yellow expectoration. Occasional attacks of palpitation were followed by paroxysmal tachycardia (pulse 130 to 140) and dyspnea. He was never able to attend school regularly.

F. H.—An uncle on his father's side and a cousin on his mother's side had fall hayfever and asthma.

He was tested for inhalants, foods, pollen, bacteria and dust, and was positive to ragweed, plus three; dust, plus two; bacteria, such as pneumococcus, influenza and tetragenus, plus two.

The patient's mother was instructed to give injections three times a week: mixed ragweed extract, 1:100,000 dilution; autogenous house dust, 1:100 dilution; autogenous vaccine, 1:50 dilution and a combination of all the bacterial proteins, 1:20 dilution.* Fall pollen was given each time, alternating with dust, autogenous vaccine and special vaccine. He called once in two weeks for a check-up to determine the dose. He was advised to abstain from school and home exercises for one year. In several months he was free from coughing, asthma, sneezing and attacks of palpitation. He was discharged after two years and is perfectly well since.

The fourth patient, a girl aged 7, was seen on November 15, 1942.

C.C.—Attacks of bronchial asthma, irregular throughout the year but worse in the fall and winter months. She had sneezing spells for one-half hour at a time, which were worse in the summer and fall. Attendance at school was irregular on account of illness with colds and asthma. Attacks of coughing, wheezing, sneezing and yellow expectoration occurred occasionally at 4 a. m.

*It is a special catarrhal vaccine prepared on request by the Sherman Laboratories, Detroit, Michigan, under the name of Dr. Sterling's Special Catarrhal Vaccine. It contains the following: Friedlander bacillus, Pneumococcus (7 types), Micrococcus catarrhalis, Streptococcus (hemolytic and non-hemolytic), Staphylococcus albus, Staphylococcus aureus, Influenza bacillus (Pfeiffer), Micrococcus tetragenous, 250 million each. 2,000 million killed bacteria per cc.

P. H.—Pertussis at age 5, measles at age 6.

F. H.—Mother has fall hayfever.

She was tested for inhalants, pollen, food, bacteria and house dust, and was found to be positive to horse dander, goat hair, duck feathers, dog hair, orris root, cat hair, cottonseed—each plus one. She was positive to fall pollen, plus three; dust, plus four and she was strongly positive to various bacteria.

The mother was instructed to use the following injections: a combination of all the inhalants, 1:10,000; mixed ragweed pollen, 1:100,000; vaccine from cultures of smears from nose, throat and sputum, 1:20; dust from their own home 1:200; all the bacterial proteins to which she gave a positive reaction, 1:20. Injections were given every day, two antigens at a time; fall pollen on Monday, Wednesday and Friday with dust and autogenous vaccine and special catarrhal alternately; inhalants on Tuesday, Thursday and Saturday with dust, vaccine and special catarrhal alternating. The antigenic dilutions could never be advanced to high concentrations. She was discharged perfectly well after two and one-half years of treatment.

The sixth patient was a boy 10 years of age from a New Jersey town, seen on September 21, 1945.

C. C.—Fall hayfever and asthma of three years' duration. He had ragweed injections for two years without any benefit. His trouble usually began in the middle of June, with dry cough, sneezing, rhinitis, nasal obstruction and conjunctivitis. He had severe attacks of asthma irregularly throughout the year.

P. H.—Croup at age of 6 months, scarlet fever at 3, pertussis at 5, mumps at 7, chickenpox and measles at 8. He was subject to colds and acute laryngitis.

F. H.—Father had attack of bronchial asthma lasting for several years. It followed the inhalation of ether while assisting a doctor in an emergency operation.

On examination the boy had large tonsils. Two years prior to this date he was admitted for a tonsillectomy. The doctor, finding him in an attack of asthma, refused to operate. Since then the patents feared tonsillectomy.

He was tested for inhalants, foods, pollen, dust and bacteria. His greatest reaction was to dust, plus four, and to bacteria, plus two, and he had a mild reaction to aspergilli, plus. There was a doubtful reaction to ragweed pollen (a concentration of 1:6,000).

We suggested treatment with pollen, dust and bacteria. However, after every attack of acute tonsillitis he would get attacks of asthma and bronchitis lasting two to three weeks at a time.

Comment: It was obvious that the tonsillar infections were responsible for recurring attacks and not the ragweed pollen as thought originally. There was also a question of how much his home in the marshlands

of New Jersey, and possibly the presence of molds and dust were responsible for his attacks because on several occasions when he was away from his home environment he was perfectly free from all his attacks.

The seventh patient was a boy aged 6 who was seen on September 3, 1943.

C. C.—Perennial hayfever and occasional asthma, worse from April to October.

He was tested for inhalants, foods, pollen, bacteria and dust, and was found to be positive to goat hair, horse dander, orris root, dog hair and goose feathers, each plus one; to beef, carrots, tomatoes, each plus one; to grass pollen, ragweed pollen, oak pollen, dust, each plus one; and to many bacteria.

He was treated with the following antigens: Inhalants—started with 1:5,000, then to 1:10,000, then to 1:20,000; ragweed, grass, oak and pollen, in separate dilutions, each starting with 1:25,000, later to 1:50,000, then 1:100,000; dust, 1:100, later 1:200; autogenous vaccine, cultures from smears of nose, throat and sputum, 1:20, then 1:50. The mother was instructed about injections. Two antigens in rotation were to be given as follows: ragweed pollen and dust on Mondays; grass and vaccines on Wednesdays; oak pollen and inhalants on Fridays.

Comment: The mother felt that every time he received injections his condition was aggravated. Therefore, the dilutions were constantly weakened until there was hardly any local reaction on intradermal testing to inhalants, pollen, dust and bacteria dilutions. The patient made satisfactory improvement in the course of several months but had irregular recurrent attacks. After one year of poor results, the father was moved to employment in a South American country. Further information of the patient's progress was lost.

Comments and Conclusion

It cannot be impressed too strongly that desensitization is the treatment "par excellence" in allergic diseases. However, it is a two-edged sword in the hands of the inexperienced. Pollen, inhalants and dust extract dilutions are powerful agents and only the trained should handle them. Occasionally, selected drugs for the use of symptomatic medication are very important to tide over the patient in occasional emergencies until he obtains the benefits from the desensitization. In spite of the great boom in the use of antihistaminic drugs, it has been a total failure in asthmatic patients. However, for its drying effect, it has a place in seasonal and perennial hayfever. Some of the antihistaminic drugs have a pharmacalogic effect similar to the nerve sedation of barbiturates, a vascular sedation of the ethylenediamine group and a drying effect similar to atropine. This latter effect is usually responsible for its claim of value for the symptomatic relief in hayfever. However, there is more to the symptomatology of hayfever than just the nasal discharges. A great objection

to the antihistaminic drugs is that in small doses they are of little or no value and in larger doses they produce unpredictable, uncontrollable bad reactions. The greatest danger in their use is that in case of toxic reaction there are no antidotes to counteract them. The author feels that ephedrine, phenobarbital, atropine and acetylsalicylic acid in properly regulated doses are much more desirable and much more effective than any antihistaminic drugs.

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Differentiation of Hypersensitive Carotid Sinus Reflex and Cardiac Arrest on the Operating Table*

FRANK H. LAHEY, M.D., *Department of Surgery*

AND

URBAN H. EVERSOLE, M.D., *Department of Anesthesiology*

When we realize the rapidity with which irreversible changes in the brain occur when it is deprived of adequate oxygen, it becomes quite evident that time is a most important consideration in the treatment of acute circulatory collapse on the operating table. This is particularly true if the collapse is the result of failure of arterial circulation due to cardiac arrest. Complete deprivation of oxygenated circulating blood to the cerebral tissues for longer than five minutes invariably results in irreversible changes. Furthermore, it is quite likely that the complete absence of blood circulation in the brain for longer than three minutes will be followed by a high percentage of patients who will not regain normal cerebral function.

Even with the closest attention on the part of the anesthesiologist and surgeon, it is inevitable that in many cases there may be a delay of from a few seconds to a minute or more in the establishment of the diagnosis of asystole. Further delay may be occasioned in making the decision as to whether it is a true cardiac arrest or a hypersensitive carotid sinus reflex particularly when the operative procedure during which the condition arises is in the neck where carotid sinus pressure can occur. This decision is extremely important since if the condition is a hypersensitive carotid sinus reflex, the pulsation in the carotids will in all probability be spontaneously restored. We have never seen an asystole due to a hypersensitive carotid sinus reflex in which pulsation was not spontaneously restored following removal of the precipitating stimulus. If, however, it is not a hypersensitive carotid sinus reflex but a true cardiac arrest, the time lost in making the decision that the emergency is not a carotid sinus reflex or treating it mistakenly as such can be of such length as to make it impossible to restore heart action or to restore it before the onset of irreversible cerebral damage. This fact emphasizes the need for quick thinking, decisive action and completely coordinated teamwork if a high percentage of these patients is to be saved.

We have on previous occasions recommended a routine procedure[2,3] in the handling of these emergencies in the operating room. The essentials of this routine are as follows: the presence in the operating room of a cardiac emergency kit consisting of a small tray containing two sterile 5 cc. syringes, two 4 inch 22 gauge "heart" needles, two 20 gauge 1½ inch needles for aspirating solutions from ampules, two 5 cc. ampules of 1% procaine, two 1 cc. ampules of 1:1000 epinephrine and two sterile files for

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opening glass ampules. It is to be noted that this kit contains two of everything, syringes, needles and ampules. Regardless of how well trained and well coordinated the surgical, anesthesia and operating room team, in an emergency of this sort there is bound to be a high degree of tension at which time the loss of one of the syringes, needles or ampules by falling on the floor and breaking could be fatal. It is for this reason that the emergency tray contains two of each.

As soon as the diagnosis of asystole is made, this emergency kit is immediately passed to the surgical (scrub) nurse who draws up 4.75 cc. of the 1% procaine plus 0.25 cc. of 1:1000 epinephrine into one of the 5 cc. syringes. She attaches the cardiac needle to the syringe and hands it to the surgeon who introduces it into one of the chambers of the heart (usually right ventricle) through the fifth intercostal space on the left. After blood can be withdrawn as evidence of the presence of the tip of the needle in the heart, the solution is injected rapidly.

During this time the anesthesiologist has started artificial respiration with 100% oxygen and has seen to it that the patient's head is lowered, and an intravenous drip of fluid is started if one is not already going.

While the surgeon is making the cardiac puncture, the nurse withdraws a second 4.75 cc. of 1% procaine and another 0.25 cc. of 1:1000 epinephrine, to be passed to the surgeon for injection through the original needle if there is no evidence of spontaneous heart beat at the end of thirty seconds following the first injection.

The surgical assistant should be preparing the left chest area for thoracotomy while the surgeon is making the cardiac injection unless either the chest or the abdominal cavity is already open. Unless there is positive evidence of spontaneous heart beats within thirty seconds after the first injection of procaine and epinephrine, cardiac massage must be instituted immediately. If the chest or abdominal cavity is open, this can be done either directly through the chest or by an incision in the diaphragm and insertion of the hand into the chest cavity. If neither the chest nor abdominal cavity is open when this emergency occurs, an incision should be made between the fourth and fifth ribs on the left and the costal cartilage of the fifth rib divided near the sternum to admit the surgeon's hand for massage. Gentle massage should be instituted at a rate of perhaps 20 to 30 times per minute until effective spontaneous heart beats are established. It has been demonstrated that careful cardiac massage which effectively but gently squeezes the heart and permits adequate refilling of the chambers between each contraction of the massaging hand produces sufficient blood flow to the brain to prevent irreparable damage for relatively long periods of time. Adams and Hand[1] have reported one patient with cardiac arrest in whom blood flow was maintained in this manner for twenty-two minutes before spontaneous heart action was established, followed by complete recovery.

A sensitive carotid sinus may produce asystole during operation. In its early phases this condition cannot be distinguished from true cardiac

arrest and only by inference can we assume the diagnosis to be a hypersensitive carotid sinus reflex. It is fairly well established that an asystole due to a true carotid sinus reflex is rarely fatal and in most instances cardiac rhythm will be spontaneously reestablished if the initiating stimulus is removed. It is rarely longer than thirty seconds to one minute before spontaneous heart action is resumed following asystole due to a hypersensitive carotid sinus.

Since the differential diagnosis between true cardiac arrest and a sensitive carotid sinus reflex is only inferential, it is obvious, as was stated earlier, that a great deal of valuable time can be lost in assuming the diagnosis to be a carotid sinus reflex should the emergency prove to be a true cardiac arrest. For a long time we have been of the opinion that any diagnosis of asystole made at the time of operation should be followed by treatment based upon the assumption that true cardiac arrest exists. Since a certain amount of time must inevitably be lost in the preparation of the solution for intracardiac injection, placing of the patient in the head-down position and preparing the chest wall for cardiac puncture, we have assumed that by the time these preparations were complete, the heart beat would be reestablished if the asystole was due to a hypersensitive carotid sinus.

The sequence of events in the course of a recent emergency (December 19, 1949) occurring in a patient on whom an operation (by F. H. L.) for second-stage removal of an esophageal diverticulum was being performed, plus the fact that it has been known for some time that some heart action may still be present even though no peripheral circulation can be detected, has led one of us (F. H. L.) to suggest and to practice in this case what seems to be an important modification in the routine treatment of these emergencies. Some time was lost owing to technical difficulties in the preparation of the solution for intracardiac infiltration and also perhaps to too great confidence that the emergency was a carotid sinus reflex because of the fact that it was known that pressure was exerted on the carotid sinus by a retractor. Because of this loss of time and while the nurse was preparing the solution for injection, the surgeon proceeded to insert the cardiac needle into the heart unattached to a syringe to determine whether or not there was cardiac contraction. As soon as the tip of the needle came in contact with the heart muscle, movement of the hub made it evident that a faint heart beat was present even though no pulse was perceptible in the exposed carotid artery. Because of this fact, the solution was not injected and after approximately thirty seconds the needle was withdrawn when a pulse could be felt in the carotid artery.

On the basis of this observation it seems reasonable to suggest that as soon as the diagnosis of asystole is made, the cardiac needle be immediately inserted into the heart. This maneuver (1) will serve to detect the presence of a faint heart beat which is not strong enough to produce a palpable peripheral pulse, and (2) may serve as sufficient stimulus to initiate the heart beat if none is present. If after the needle is inserted it is evident

that no heart beat is present, the routine treatment for cardiac arrest may be proceeded with as previously described. While the introduction of a needle through the chest wall into the heart is something that should not be taken lightly, the circumstances justify it. One sole possible complication must be kept in mind. Cases have been reported following this procedure of accumulation of enough blood in the pericardial sac to produce cardiac tamponade. Multiple punctures are more likely to bring about this condition and it is possible, we believe from our own experience with heart needling, to obtain the information one desires with a single introduction of the heart needle into the cardiac wall.

When we realize the gravity of this emergency and how much is to be gained by adequate treatment and how much is to be lost by inadequate treatment and loss of time, it becomes evident that if any large percentage of these patients is to be saved there must be a well-established plan of treatment which can be put into immediate action. There must be no hesitancy in the adoption of what may seem radical procedures, and there must be the utmost in coordination and teamwork by everyone concerned with the treatment.

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BOOK REVIEW

PHYSIOLOGY OF HEAT REGULATION AND THE SCIENCE OF CLOTHING, edited by L. H. Newburgh, M.D., W. B. Saunders Company, 1949.

Man is in possession of an elaborate and complicated mechanism which keeps his internal organs at a nearly constant temperature. This implies that heat is lost by the organism at the rate at which it is produced. The heat is carried from the warmer interior to the cooler body surfaces by the blood and to a lesser extent by conduction across the tissues. It is dissipated from these surfaces by radiation, convection and evaporation of water. The part played by each of these factors and their integration had already been adequately described for conditions which in retrospect could justifiably be thought of as moderate or median states.

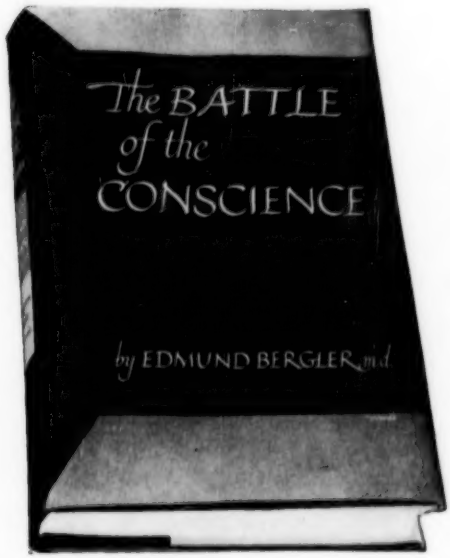
The contributors, while recognizing that this information formed a fundamental basis for their future studies, concentrated upon discovering the extreme limits of this mechanism. They sought to determine the maximal rate of heat production that can be endured in the hottest and most humid environments and at the other extreme, how long survival is possible in cold water and very cold air, to what extent and for how long man can survive in the cold by increasing his production of internal heat through muscular activity.

It is common knowledge that the loss of heat is lessened by clothing, but no one knows how much protection could be obtained by this means. The clothes worn by man are dictated by custom and fashion. Little thought had been given to the protective possibilities inherent in the raw fibers and the various ways they can be combined into cloth. The authors assumed a study of the physical qualities of fabrics would form a basis for devising new textiles that were better heat barriers than any available. They examined the clothing and shelter customs of groups that had survived in the extremes of climate.

This book attempts to describe the responses of heat regulatory mechanism to the whole range of climatic conditions encountered on the earth's surface. It emphasizes the maximal capacity of the mechanism to preserve life under the most adverse conditions and establishes the limits of this capacity. It shows how far the rigours of unfavorable environments can be mitigated by scientifically designed protective coverings. Studies are being continued by both military and civilian groups.

The Battle of the Conscience

By
EDMUND BERGLER, M.D.



"This book is documented with a wealth of clinical material culled from the long practice of the author. As an example of some of the current ramifications of Freudian theory it is extremely interesting and warrants careful study."

Connecticut State Journal of Medicine

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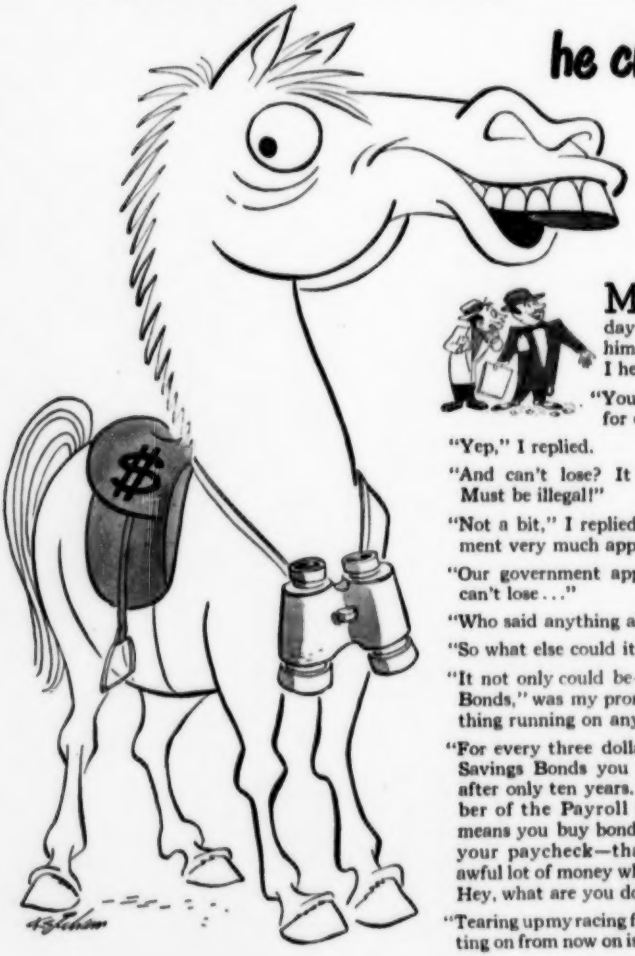
"There's no such animal," he cried!



MY FRIEND and I were picking the ponies one day when I started telling him about *a sure thing* I heard about.

"You say it pays four bucks for every three?" he asked.

"Yep," I replied.

"And can't lose? It *automatically* wins? Must be illegal!"

"Not a bit," I replied. "In fact, the government very much approves..."

"Our government approves of a horse who can't lose..."

"Who said anything about a horse?" I asked.

"So what else could it be but a horse...?"

"It not only could be—but is—U. S. Savings Bonds," was my prompt reply. "The surest thing running on any track today.

"For every three dollars you invest in U. S. Savings Bonds you get four dollars back after only ten years. And if you're a member of the Payroll Savings Plan—which means you buy bonds *automatically* from your paycheck—that can amount to an awful lot of money when you're not looking. Hey, what are you doing?"

"Tearing up my racing form! The horse I'm betting on from now on is U. S. Savings Bonds."

Automatic saving is <u>sure</u> saving—U.S. Savings Bonds

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